IN THE SPECIFICATION:

On page 3, please replace the paragraph spanning lines 15-22 with the following amended paragraph:

> The present invention thus relates to a DNA sequence of the 5' flanking region of the 5-HT1A receptor gene, from about -3438 to about -393 (SEQ ID NO:1), wherein said sequence contains a mutation which results in an inhibition of protein-DNA interactions. The partial wild type sequence of the human 5-HT1A receptor gene is deposited in Genbank and has been published by Parks ~~ans~~ and Shenk (1996). More specifically this invention relates to a AND sequence comprising a polymorphic C-G change at position -1017 (position 2422 of SEQ ID NO:1) of the 5-HT1A receptor gene and to a 31 base pair region flanking the -1017 locus.

On page 6, please replace the paragraph spanning lines 4-13 with the following amended paragraph:

> **FIGURE 6** shows the association of nuclear proteins with the polymorphic site of the 5-HT1A receptor gene. Gel mobility shift assay was done using nuclear extracts prepared from RN46A cells. The specific 31-bp probe spans the palindrome where the polymorphic point mutation in depressed patients has been found at -1017 bp (position 2422 of SEQ ID NO:1) from the initial ATG codon of the human 5-HT1A promoter sequence, and was present in all samples. Nuclear extract, 100-fold molar excess of unlabeled specific 31-bp oligonucleotide of unrelated sequence were added to the incubation as indicated. A specific shifted complex is indicated by the arrowhead.

On page 7, please replace the paragraph spanning lines 17-31 with the following amended paragraph:

> In one embodiment of the present invention a C-G change at -1017 bp (position 2422 of SEQ ID NO:1) was identified. According to the present invention, the occurrence of G at -1017 bp was found to correlate with patients with mental

illness. In a population of depressed patients 80% were either homozygous or heterozygous for this change; 30% of the patients were homozygous for this change. Prior to the present invention there was no evidence for a clear genetic association with a particular mental illness. From the results of the present invention, the identification of a homozygous C-G polymorphism, which strongly correlated with depressed patients and absent in normals, provides evidence of the use of this polymorphism as a genetic marker for mental illness. Increasingly, PCR-based gene detection is being used in prognostic and diagnostic evaluation of patients, and in criminological identification and characterization. For example, genetic testing of children of affected adults may allow for counseling or early treatment prior to development of an episode of major depression. Furthermore, as data accumulates it may be possible to correlate the genetic change with properties such as severity or drug treatment response.